

## Effects of Vestibular Loss on Orthostatic Responses to Tilts in the Pitch Plane

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The purpose of this study was to determine the extent to which vestibular loss might impair orthostatic responses to passive tilts in the pitch plane in human subjects. Data were obtained from six subjects having chronic bilateral vestibular loss and six healthy individuals matched for age, gender, and body mass index. Vestibular loss was assessed with a comprehensive battery including dynamic posturography, vestibulo-ocular and optokinetic reflexes, vestibular evoked myogenic potentials, and ocular counterrolling. Head up tilt tests were conducted using a motorized two-axis table that allowed subjects to be tilted in the pitch plane from either a supine or prone body orientation at a slow rate (8 deg/s). The sessions consisted of three tilts, each consisting of 20 min rest in a horizontal position, tilt to 80 deg upright for 10 min, and then return to the horizontal position for 5 min. The tilts were performed in darkness (supine and prone) or in light (supine only). Background music was used to mask auditory orientation cues.

Autonomic measurements included beat-to-beat recordings of blood pressure (Finapres), heart rate (ECG), cerebral blood flow velocity in the middle cerebral artery (transcranial Doppler), end tidal CO<sub>2</sub>, respiratory rate and volume (Respritrace), and stroke volume (impedance cardiography). For both patients and control subjects, cerebral blood flow appeared to exhibit the most rapid adjustment following transient changes in posture. Outside of a greater cerebral hypoperfusion in patients during the later stages of tilt, responses did not differ dramatically between the vestibular loss and control subjects, or between tilts performed in light and dark room conditions. Thus, with the exception of cerebrovascular regulation, we conclude that orthostatic responses during slow postural tilts are not substantially impaired in humans following chronic loss of vestibular function, a result that might reflect compensation by nonvisual graviceptor inputs (e.g., somatosensory) or other circulatory reflex mechanisms.

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